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Finnegan, Henderson, Farabow,
Garrett & Dunner, L.L.P.
1300 I Street, N.W.
Washington, DC 20005-3315

EXAMINER	
CHEN, STACY BROWN	
ART UNIT	**PAPER NUMBER**
1648	

DATE MAILED: 12/28/2004

Please find below and/or attached an Office communication concerning this application or proceeding.

Office Action Summary	Application No.	Applicant(s)	
	10/700,491	AMARA ET AL.	
	Examiner	Art Unit	
	Stacy B Chen	1648	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *15 September 2004*.

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1-71* is/are pending in the application.

4a) Of the above claim(s) *3,7,8,21,22,31 and 42-71* is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *1,2,4-6,9-20,23-30 and 32-41* is/are rejected.

7)☒ Claim(s) *1,30 and 38-41* is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☒ The specification is objected to by the Examiner.

10)☒ The drawing(s) filed on *05 November 2003* is/are: a)☒ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☒ Information Disclosure Statement(s) (PTO-1449 or PTO/SB/08) Paper No(s)/Mail Date *5.26.94*.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____ .
5) ☐ Notice of Informal Patent Application (PTO-152)
6) ☐ Other: _____.

DETAILED ACTION

1. In the response filed September 15, 2004, Applicant's election with traverse of Group II, HIV, is acknowledged. Applicant's arguments have been carefully considered. Applicant argues that the restriction requirement improperly limits the scope of the claims by requiring restriction between Groups I-IV. For example, Group I is drawn to a method of preventing or treating a mammal with a DC-SIGN modulator that is a derivative of an effector molecule. However, claim 1 of Group I is drawn to a method that uses a generic DC-SIGN modulator, whereas claim 3 recites that the modulator is a derivative of an effector molecule. Therefore, Applicant asserts that the Office has improperly limited the claims to certain embodiments in the dependent claims.

In response, the Office regrets any confusion regarding the claim groupings. The Office had no intention of limiting the scope of claims that recite generic embodiments. The reason that claims 1, 2, 9-14 and 23-30 were included in each of Groups I-IV is that they are *linking claims*. Claims 38-40 should have also been included as linking claims between Groups I-IV. The next paragraph details more clearly what was originally intended by the restriction requirement.

Claims 1, 2, 9-14, 23-30 and 38-40 link inventions I-IV. The restriction requirement between the linked inventions is subject to the nonallowance of the linking claim(s), claims 1, 2, 9-14, 23-30 and 38-40. The inventions of Groups I-IV (reflective of the original groupings without the linking claims) are as follows:

- Group I (derivative of an effector molecule): claim 3
- Group II (antibody): claims 4-6, 15-20 and 32-37
- Group III (mannosylated molecule): claims 7, 8, 21 and 22

- Group IV (recombinant protein): claim 31

Upon the allowance of the linking claim(s), the restriction requirement as to the linked inventions shall be withdrawn and any claim(s) depending from or otherwise including all the limitations of the allowable linking claim(s) will be entitled to examination in the instant application. Applicant(s) are advised that if any such claim(s) depending from or including all the limitations of the allowable linking claim(s) is/are presented in a continuation or divisional application, the claims of the continuation or divisional application may be subject to provisional statutory and/or nonstatutory double patenting rejections over the claims of the instant application. Where a restriction requirement is withdrawn, the provisions of 35 U.S.C. 121 are no longer applicable. *In re Ziegler*, 44 F.2d 1211, 1215, 170 USPQ 129, 131-32 (CCPA 1971). See also MPEP § 804.01.

Regarding the restriction requirement, Applicant also requests clarification whether claims 38-41 were to be restricted to HIV or SIV, or whether all of claims 1-41 were also to be restricted to HIV or SIV. In response, only claims 38-40 are restricted to either HIV or SIV. Claim 41 will be examined if HIV is elected since claim 41 is drawn to an embodiment of HIV. Applicant also argues that the Office has not demonstrated a serious burden of examination of HIV and SIV. In response, the Office indicated that searches for HIV and SIV are divergent. HIV and SIV are different immunodeficiency viruses having different viral pathologies. While they are both immunodeficiency viruses and classified similarly, literature that speaks to HIV will not necessarily discuss SIV and vice versa. Further, methods of treating HIV and SIV are not one and the same. SIV is the closest animal model of HIV infection, however, SIV is not an

accepted animal model for predicting HIV treatment in humans. Therefore, searching methods of treating/preventing HIV and SIV places a serious burden on the Office.

Therefore, the restriction requirement as clarified above is deemed proper and made FINAL. Claims 1-71 are pending. Claims 3, 7, 8, 21, 22, 31 and 42-71 are withdrawn from consideration, being drawn to non-elected inventions. Claims 1, 2, 4-6, 9-20, 23-30 and 32-41 are under examination.

Information Disclosure Statement

2. The information disclosure statement filed May 26, 2004 is acknowledged and a copy is attached to this Office action. Note that Patent Application numbers 10/464,531 and 10/700,507 have been considered but will not be printed on the face of the file of any patent that will issue.

Claim Objections

3. Claims 1 and 38 are objected to for failing to spell out the acronyms DC-SIGN and HIV, respectively, at their first occurrence. Claims 38, 39 and depending claims 40 and 41 are objected to for reciting non-elected inventions which are not eligible for rejoinder at any point in future prosecution in this application.

Claim Rejections - 35 USC § 112

4. The following is a quotation of the first paragraph of 35 U.S.C. 112:

> The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

Claims 23-30 and 32-37 are rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for a method of treating a flavivirus infection or Dengue virus infection, does not reasonably provide enablement for preventing such infections. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make or use the invention commensurate in scope with these claims. The breadth of the claims is unreasonable, encompassing prevention of any flavivirus infection or dengue virus infection with a modulator/blocker, particularly with antibodies (passive immunization). The family of flaviviruses includes for example, Hepatitis C (HCV), Yellow fever, Dengue, Japanese encephalitis, tick-borne encephalitis, pestivirus, border disease and classical swine fever virus. The nature of the invention encompasses prevention of any of these viruses of the flavivirus family by blocking entry of these viruses into cells by preventing DC-SIGN from binding to the viruses. The state of the art shows that there are no vaccines for flaviviruses. For example, the Centers for Disease Control (CDC)[i] reports that there is no vaccine for Dengue virus and that efficacy trials in humans have yet to be initiated as of the year 2003 (see website printout, page 4, "Future Outlook" section). Leyssen *et al.* (*Clin. Microbiol. Rev.* 2000, 13(1):67-82, herein, "Leyssen") confirms that there are no vaccines or treatments for Dengue virus (page 72, column 1, first full paragraph). Current treatment for HCV is ribavirin, but with limited results and no protection (Leysson, page 72, top incomplete paragraph). Leyssen teaches that little is known about flavivirus entry and cell receptor (page 73, columns 1 and 2, bridging paragraph). Leyssen also discloses that because of "the genetic and serological heterogeneity of HCV, the development of effective vaccines will be difficult and is not expected to occur soon" (page 76, column 2, last paragraph). Regarding Dengue virus, Japanese

encephalitis virus and tick-borne encephalitis virus, Leyssen discloses that there are no drugs yet available (page 76, column 2, last paragraph). Men *et al.* (*J. Virology*, 2004, 78(9):4665-4674) also confirms that there are no vaccines for Dengue virus (abstract). The level of skill in the art is high. The level of predictability in the art is low, evidenced by the above discussion of the state of the art. The specification does not provide guidance or working examples of prevention of flavivirus infection in an acceptable animal model. It would require undue experimentation to use the claimed invention to prevent flavivirus infections. Therefore, the claims are not enabled for their full scope.

5. Claims 38-41 are rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for a method of treating human immunodeficiency virus type 1 (HIV-1) infection, does not reasonably provide enablement for preventing all types of HIV infections. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to use the invention commensurate in scope with these claims. The breadth of the claims is unreasonable, encompassing prevention of HIV infection *in vivo*. The nature of the invention is the prevention of HIV by inhibiting HIV gp120 from binding to cells expressing DC-SIGN, thereby preventing viral entry and infection. The state of the art shows *treatments* that reduce viral entry by inhibiting gp120 from binding CD4 receptors on T cells. Greene (*Nature Immunology*, 2004, 5(9):867-871) discloses that HIV attachment inhibitors, which block gp120 from binding to the CD4 receptor, are effective for reducing viral load (Greene, page 867, second and third column). However, the state of the art also teaches that there are no preventative treatments for HIV. Desrosiers (*Nature Medicine*, March 2004,

10(3):221-223) teaches that the natural immune response to HIV-1, humoral and T-cell responses, are ineffective due to antigenic variation/mutation, resistance to antibody-mediated neutralization, down regulation of major histocompatibility class I molecules from the surface of infected cells and destruction of $CD4^+$ T helper cells (page 221, cols. 1-2). Vaccine strategies for protecting rhesus monkeys from SIV, the closest animal model to HIV-1 infection in humans, yet an unacceptable model, has been unsuccessful. The variability of sequences among HIV-1 isolates is enormous, making it impossible to date to construct epitopes that are neutralizing across HIV-1 isolates. Vaccine trials using peptide vaccines against HIV-1 using recombinant gp120 have failed to induce protective immunity (page 222, col. 1). Feinberg *et al.* (*Nature Medicine*, March 2002, 8(3):207-210, herein, "Feinberg") discloses that there are no acceptable animal models that reflect the actual biological pathology of HIV-1 in humans. Rhesus monkeys cannot be infected with HIV-1, so chimeric constructs of HIV and SIV are used. Unfortunately, this model of infection, while useful, does not reflect HIV-1 infection/pathology in humans in many respects. The main drawback of this model is that promising responses in the model are not direct translations into success in humans. For example, the rapid $CD4^+$ T helper cell depletion in the animal model is due to the nature of viral entry, which primarily uses the CXCR4 viral coreceptor. This is not consistent with the majority of HIV-1 viruses transmitted between humans which uses the CCR5 coreceptor, resulting in slower depletion of $CD4^+$ T helper cells in humans. The use of different coreceptors is an important consideration for designing vaccines in humans (page 208). Further, the SHIV model is sensitive to autologous neutralizing antibodies, whereas most primary HIV-1 isolates resist antibody neutralization (page 208-209, bridging paragraph). The level of skill in the art is high. The level of predictability in

the art regarding HIV treatments *in vivo* is low, evidenced by the above discussion. There is no guidance in the specification or working examples demonstrating prevention of HIV in an acceptable animal model. It would require undue experimentation to use the claimed inventive method to prevent HIV infection. Therefore, the claims are not enabled for their full scope.

6. The following is a quotation of the second paragraph of 35 U.S.C. 112:

> The specification shall conclude with one or more claims particularly pointing out and distinctly claiming the subject matter which the applicant regards as his invention.

Claims 1, 2, 4-6, 9-20, 23-30 and 32-41 are rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention. The claims recite that the DC-SIGN modulator is administered in an amount sufficient to "substantially" modulate/inhibit binding of the effector molecule to DC-SIGN. It is unclear what the metes and bounds are of the amount of modulator required to substantially modulate/inhibit binding. The specification does not clearly define what amount results in a substantial modulation or inhibition of binding. There should be endpoints indicating that a substantial level of modulation/inhibition has been achieved.

Claim Rejections - 35 USC § 102

7. The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

> A person shall be entitled to a patent unless –
>
> (a) the invention was known or used by others in this country, or patented or described in a printed publication in this or a foreign country, before the invention thereof by the applicant for a patent.

(b) the invention was patented or described in a printed publication in this or a foreign country or in public use or on sale in this country, more than one year prior to the date of application for patent in the United States.

Claims 1, 2, 4-6, 9-20, 38, 39 and 41 are rejected under 35 U.S.C. 102(a) as being anticipated by Littman *et al.* (US Patent 6,391, 567 B1, herein, "Littman '567"). The claims are drawn to a method of treating a disease of a mammal, wherein at least one symptom of the disease is mediated at least in part by the binding of an effector molecule to a DC-SIGN receptor of the mammal to be treated, wherein the method comprises administering to the mammal an amount of a DC-SIGN modulator sufficient to substantially modulate the biding of the effector molecule to the DC-SIGN receptor to thereby treat the disease. (Note that the claims also recite prevention of disease, however, the claims are only enabled for treatment of disease and are therefore presently only treated for their enabled embodiment.) Specifically, the DC-SIGN modulator is a blocker that inhibits the binding of the effector molecule to the DC-SIGN receptor. The DC-SIGN blocker is an antibody, such as a monoclonal antibody or humanized monoclonal antibody. The antibody specifically binds DC-SIGN, or in other embodiments, binds the viral effector molecule. More specifically, the disease being treated is a viral disease, wherein the viral effector molecule is a molecular constituent of the viral envelope, such as envelope glycoprotein. The viral disease can be HIV infection in a human.

Littman '567 discloses a method for treating HIV infection in humans comprising administering humanized monoclonal antibodies (modulator/blocker) that bind DC-SIGN on dendritic cells. The binding of DC-SIGN prevents HIV gp120 (binding moiety of viral effector molecule) from interacting with DC-SIGN. Also disclosed are antibodies that bind to the viral effector molecule, such as antibodies to gp120. (See col. 10, lines 56-65, col. 20, lines 34-67, and col. 25, lines 25-56.) Therefore, the claims are anticipated by Littman '567.

8. Claims 1, 2, 4, 6, 9-17, 38, 39 and 41 are rejected under 35 U.S.C. 102(b) as being anticipated by Littman *et al.* (WO 01/64752 A2). The claims are drawn to a method of treating a disease of a mammal, wherein at least one symptom of the disease is mediated at least in part by the binding of an effector molecule to a DC-SIGN receptor of the mammal to be treated, wherein the method comprises administering to the mammal an amount of a DC-SIGN modulator sufficient to substantially modulate the biding of the effector molecule to the DC-SIGN receptor to thereby treat the disease. (Note that the claims also recite prevention of disease, however, the claims are only enabled for treatment of disease and are therefore presently only treated for their enabled embodiment.) Specifically, the DC-SIGN modulator is a blocker that inhibits the binding of the effector molecule to the DC-SIGN receptor. "DC-SIGN receptor" and "DC-SIGN" are synonymous terms because DC-SIGN is a receptor. The DC-SIGN blocker is an antibody, such as a monoclonal antibody or humanized monoclonal antibody. The antibody specifically binds the viral effector molecule. More specifically, the disease being treated is a viral disease, wherein the viral effector molecule is a molecular constituent of the viral envelope, such as envelope glycoprotein. The viral disease can be HIV infection in a human.

Littman *et al.* discloses antibodies (modulator/blocker) specific for the antigenic fragment of gp120 (envelope subunit protein of HIV and binding moiety of viral effector molecule) that inhibits DC-SIGN on dendritic cells from interacting with gp120. Also disclosed are methods of treating HIV infection by administering antibodies that bind to gp120, thereby inhibiting binding of gp120 to DC-SIGN. The antibodies can be humanized, monoclonal antibodies. (See Littman *et al.*, page 5, pages 5-6, bridging paragraph, and claims 1-4.)

9. Claims 1, 2, 4, 5, 9-12, 15-18, 38, 39 and 41 are rejected under 35 U.S.C. 102(b) as being anticipated by Figdor *et al.* (EP 1046651 A1, herein, "Figdor"). The claims are drawn to a method of treating a disease of a mammal, wherein at least one symptom of the disease is mediated at least in part by the binding of an effector molecule to a DC-SIGN receptor of the mammal to be treated, wherein the method comprises administering to the mammal an amount of a DC-SIGN modulator sufficient to substantially modulate the biding of the effector molecule to the DC-SIGN receptor to thereby treat the disease. (Note that the claims also recite prevention of disease, however, the claims are only enabled for treatment of disease and are therefore presently only treated for their enabled embodiment.) Specifically, the DC-SIGN modulator is a blocker that inhibits the binding of the effector molecule to the DC-SIGN receptor. The DC-SIGN blocker is an antibody, such as a monoclonal antibody or humanized monoclonal antibody. The antibody specifically binds DC-SIGN. More specifically, the disease being treated is a viral disease, wherein the viral effector molecule is a molecular constituent of the viral envelope, such as envelope glycoprotein. The viral disease can be HIV infection in a human.

Figdor discloses a method for treating HIV infection in humans comprising administering humanized monoclonal antibodies (modulator/blocker) that bind DC-SIGN on dendritic cells. The binding of DC-SIGN prevents HIV gp120 (binding moiety of viral effector molecule) from interacting with DC-SIGN (page 4, [0040], page 5, [0046] and page 7, [0070]-[0071]). Therefore, the claims are anticipated by Figdor.

10. Claims 1, 2, 4, 6, 9-16, 19, 20, 23, 25-30, 32-33, 36 and 37 are rejected under 35 U.S.C. 102(b) as being anticipated by Brandriss *et al.* (*J. Gen. Virology*, 1986, 67:229-234, herein, "Brandriss"). The claims are drawn to a method of treating a disease of a mammal, wherein at least one symptom of the disease is mediated at least in part by the binding of an effector molecule to a DC-SIGN receptor of the mammal to be treated, wherein the method comprises administering to the mammal an amount of a DC-SIGN modulator sufficient to substantially modulate the biding of the effector molecule to the DC-SIGN receptor to thereby treat the disease. (Note that the claims also recite prevention of disease, however, the claims are only enabled for treatment of disease and are therefore presently only treated for their enabled embodiment.) Specifically, the DC-SIGN modulator is a blocker that inhibits the binding of the effector molecule to the DC-SIGN receptor. The DC-SIGN blocker is an antibody, such as a monoclonal antibody or humanized monoclonal antibody. The antibody specifically binds the viral effector molecule. More specifically, the disease being treated is a viral disease, Dengue, wherein the viral effector molecule is a molecular constituent of the viral envelope, such as envelope glycoprotein.

Brandriss discloses monoclonal antibodies against the E glycoprotein of Dengue virus administered to mice prior to and subsequent to challenge with Dengue virus (page 230, last paragraph and Table 1). While Brandriss does not disclose the DC-SIGN blocker activity of the antibody to Dengue E glycoprotein, the act of administering the antibody to mice reads on the claimed method of treating a disease of a mammal by administering a DC-SIGN blocker. Although the antibody is not called a "DC-SIGN blocker", the antibody's identity remains the same as that claimed by Applicant. Brandriss' antibody binds to Dengue glycoprotein E and

inherently affects the binding of glycoprotein E with DC-SIGN on the cells of the mice. Therefore, the claims are anticipated by Brandriss.

Claim Rejections - 35 USC § 103

11. The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

> (a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

Claims 24 and 34 are rejected under 35 U.S.C. 103(a) as being unpatentable over Brandriss as applied to claims 1, 2, 4, 6, 9-16, 19, 20, 23, 25-30, 32-33, 36 and 37 above, and further in view of Hoogenboom *et al.* (US Patent 5,565,332, herein, "Hoogenboom"). The claims are drawn to a method of treating flavivirus infection in a mammal comprising administering a DC-SIGN blocker, such as a humanized antibody to a human. Specifically, the flavivirus is Dengue virus. Brandriss teaches the administration to mice of monoclonal antibodies that bind to Dengue glycoprotein E. Brandriss does not teach a humanized antibody to be administered to humans to treat Dengue virus infection.

At the time of the instant invention, it would have been obvious to humanize Brandriss' antibodies. One would have been motivated to administer anti-E antibodies to humans because of the long felt need in the art for a Dengue vaccine, evidenced by Leyssen (cited above) and Men *et al.* (cited above) which teach that there are no vaccines for Dengue virus despite a great need for vaccines (abstract). One would have been motivated to humanize the rodent antibodies because of the undesirable immune response caused by administering rodent antibodies to

humans, taught by Hoogenboom. Hoogenboom discloses that humanized antibodies have been made to several viruses (col. 1, lines 8-56). Humanized antibodies deter unwanted immune responses in humans. One would have had a reasonable expectation of success that the antibodies would have successfully been humanized because Hoogenboom's antibodies were humanized, as were others (col. 1, lines 24-56). Therefore, the claims would have obvious over Brandriss in view of Hoogenboom.

This application currently names joint inventors. In considering patentability of the claims under 35 U.S.C. 103(a), the examiner presumes that the subject matter of the various claims was commonly owned at the time any inventions covered therein were made absent any evidence to the contrary. Applicant is advised of the obligation under 37 CFR 1.56 to point out the inventor and invention dates of each claim that was not commonly owned at the time a later invention was made in order for the examiner to consider the applicability of 35 U.S.C. 103(c) and potential 35 U.S.C. 102(e), (f) or (g) prior art under 35 U.S.C. 103(a).

Double Patenting

12. A rejection based on double patenting of the "same invention" type finds its support in the language of 35 U.S.C. 101 which states that "whoever invents or discovers any new and useful process ... may obtain a patent therefor ..." (Emphasis added). Thus, the term "same invention," in this context, means an invention drawn to identical subject matter. See *Miller v. Eagle Mfg. Co.*, 151 U.S. 186 (1894); *In re Ockert*, 245 F.2d 467, 114 USPQ 330 (CCPA 1957); and *In re Vogel*, 422 F.2d 438, 164 USPQ 619 (CCPA 1970).

A statutory type (35 U.S.C. 101) double patenting rejection can be overcome by canceling or amending the conflicting claims so they are no longer coextensive in scope. The filing of a terminal disclaimer cannot overcome a double patenting rejection based upon 35 U.S.C. 101.

Claims 1, 2, 9-14, 38, 39 and 41 are provisionally rejected under 35 U.S.C. 101 as claiming the same invention as that of claims 1, 2, 9-14, 39, 41 and 42 of copending Application No. 10/700,507. This is a provisional double patenting rejection since the conflicting claims have not in fact been patented.

Conclusion

13. No claim is allowed. The subject matter of claim 35, drawn to a method of treating flavivirus infection using an antibody that binds to DC-SIGN is not taught or suggested in the prior art of record. The prior art made of record and not relied upon is considered pertinent to applicant's disclosure. Tassaneetrithep *et al.* (*J. Exper. Med.* 2003, 197(7):823-829) discloses that DC-SIGN (also known as CD209) mediates Dengue virus infection of human dendritic cells and that anti-DC-SIGN antibodies may be considered for designing therapies that block dengue infection at the entry (envelope) level (abstract and page 828).

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR

system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free).

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Stacy B. Chen whose telephone number is 571-272-0896. The examiner can normally be reached on M-F (7:00-4:30). If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, James C. Housel can be reached on 571-272-0902. The fax phone number for the organization where this application or proceeding is assigned is 703-872-9306.

Stacy B. Chen
December 21, 2004

[i] CDC Dengue Fever Home Page, http://www.cdc.gov/ncidod/dvbid/dengue/index.htm